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# THE OBESITY PROBLEM: IS IT A STATE *IN* MIND?

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**Index Terms**: *Obesity; Control; Mental Models; Decision Making; System Dynamics*

# THE OBESITY PROBLEM: IS IT A STATE *IN* MIND?


## Abstract

The growing obesity problem is addressed from a decision-theoretic perspective. In managing their health (and their bodies), people are viewed as decision makers who are managing a truly complex and dynamic system – the human body. The question is how effective the mental models that people rely on are for such a task? Specifically, our objective is twofold. First, to demonstrate the deficiencies in people's mental models and the impacts they have on the treatment and prevention of obesity. Second, to demonstrate the utility of *System Dynamics* modeling tools to address these deficiencies through learning and decision support.

A holistic system dynamics computer model is presented that integrates the processes of human metabolism, hormonal regulation, body composition, nutrition, and physical activity. The model serves as a laboratory tool for controlled experimentation to gain insight into the dynamics of body weight regulation.

Three simulation experiments are conducted. The results of the first two experiments demonstrate how the regulation of energy balance is significantly influenced by *involuntary* metabolic/physiologic adaptations that regulate both energy expenditure and energy intake. Ignoring such adaptations leads to spurious predictions of treatment outcomes.

A significant impediment to obesity prevention, on the other hand, is an "automatic weight control" mentality that instills in people a false sense of invulnerability. The results of the third experiment demonstrate and quantify the *asymmetry* of the body's weight-regulation processes. The results underscore the need for replacing the passive model of involuntary/automatic weight maintenance with an assertive model of cognitive control to proactively resist the obesifying aspects of the current environment.

Simulation-based *Microworlds* are proposed as laboratory tools for double-loop learning to induce deep change in people's mental models and create more effective decision rules and strategies.



**Index Terms**: *Obesity; Control; Mental Models; Decision Making; System Dynamics*


## I. INTRODUCTION

The number of Americans who are obese is at its highest level ever recorded [31]. Most obese individuals attempting to lose weight do so themselves without seeking professional help [9]. It has been estimated that Americans spend approximately $33 billion annually on losing weight, an amount that is enough to buy every item and service produced in such countries as Chile, Croatia, or Nigeria [24, p. 6]. Of concern is that the great effort invested in preventing and treating obesity is often wasted on ineffective, sometimes even harmful practices that extend beyond the merely unwise to the Faustian. After years of little change, sales of diet pills and supplements have more than quadrupled since 1996, invigorated by what appears to be a broad retreat from weight loss regimens that require sweat and sacrifice [35]. The surging demand for a weight loss "silver bullet" has fueled a rapid rise in fraudulent weight-loss schemes, including: slimming soaps that slough off fat in the shower; miracle pills that get rid of excess pounds without dieting or exercise; plastic earplugs that curb the appetite; and even a glittering ring called Fat-Be-Gone that when slipped on a finger trims hips, buttocks and thighs [35].

In this paper, the growing obesity problem is addressed from a decision-theoretic perspective. In managing their health (and their bodies), people are viewed as decision makers who are managing a truly complex and dynamic system – the human body. The question is how effective are the mental models that people rely on for such a task?

## II. CONCEPTUAL BACKGROUND

People form mental models (or conceptualizations) of themselves, others, the environment, and the things with which they interact through experience, training, and instruction. These cognitive maps are essential in helping people make sense of their experiences and in predicting the outcomes of their actions [21, p. 39]. Mental models have been described as,

> … deeply ingrained assumptions, generalizations, or even pictures or images that influence how we understand the world and how we take action… (Mental models) are not simply repositories for past learning: they are also the basis for interpretations of what is currently happening and they strongly influence how the person will act in response [3].

Mental models are viewed as indispensable to human information processing because they organize knowledge in simple, robust and parsimonious ways, in a world awash with information of staggering complexity. People base their models on whatever knowledge they have, real or imaginary, naïve or sophisticated [21, p. 38]. Once formed, mental models determine not only how people make sense of the world, they also shape how they act, determine how environmental stimuli will be interpreted and incorporated or synthesized, and even whether or not cues will be noticed and used.

For decades, a dominant position in the literature has maintained that well-adjusted individuals possess relatively accurate mental models (perceptions) of themselves, their capacity to control important events in their lives, and their future [29]. Despite its plausibility, this viewpoint is increasingly difficult to maintain. A great deal of research in social, personality, clinical, and developmental psychology has revealed that people's actual inferential work and decision making looks little like these normative models. That mental models are only simplified abstractions of the experienced world, and are often incomplete, reflecting a world that is only partially understood [3]. Furthermore, human information processing has been found to be affected by needs and emotions, and too often governed not by normative principles but by cognitive shortcuts and biases [22]. For example, experiments over a wide variety of tasks reveal that prior expectations and self-serving

interpretations weigh heavily into the social judgment process, and that people's predictions of what will occur correspond closely to what they would like to see happen or to what is socially desirable rather than to what is objectively likely [29].

One domain in which most individuals' perceptions appear to be less than realistic concerns beliefs about *personal control.* Many theorists have maintained that a sense of personal control is integral to the self-concept and self-esteem. Research evidence, however, suggests that people's beliefs in personal control are sometimes greater than can be justified [29]. For example, studies have shown that many cancer patients professed a high degree of control over the future course of their cancer, even when their prognoses were poor and recurrence was a virtual certainty [30]. Laboratory evidence shows that people are vulnerable to imputing control even to random events. In a series of studies adopting gambling formats, Langer [18] and her associates found that people often act as if they have control in situations that are actually determined by chance. Langer [18] coined the phrase "illusion of control" to refer to people's systematic tendency to assess subjective probabilities of success that are higher than objective circumstances would warrant.

Research by Taylor and his colleagues ([29], [30]) suggests that exaggerated perceptions of control are not only characteristic of normal human thought, but, paradoxically, may be adaptive for mental health and well-being. Exaggerated perception of control was shown, for example, to help people approach potential stressors or threats with the confidence that they can exercise some control over them. Such an outlook enhances performance accomplishments, reduces stress, and lowers vulnerability to depression. In health-related domains, individuals with higher levels of perceived control were found to take greater responsibility for meeting their health needs, were more likely to comply with medical regimens involving lifestyle/habit changes and to maintain the new habit over time [12].

There are risks, however. One consequence of assuming more control than actually exists is that people may set unrealistically high goals that will increase the likelihood or costliness of failure. Furthermore, faith in one's capacity to master situations may lead people to persevere at tasks that may, in fact, be uncontrollable [29]. Excessive persistence in futile endeavors increases the costs (e.g., time, effort, and money) that accompany the failure. The end result is often increased frustration and other emotional costs. In the health domain, exaggerated perceptions of control create an atmosphere in which bad health can be perceived as a personal or moral failing… and the burden of illness is joined by the burden of guilt [8].

In conclusion, the overriding implication that we can draw from the analysis of this literature is that the mentally healthy appears to have the enviable capacity to distort reality in a direction that enhances self-esteem, maintains beliefs in personal efficacy, and promotes an optimistic view of the future [8]. In this sense, the capacity to develop and maintain positive illusions may help make each individual's world a warmer place in which to live. On the other hand, there are risks – especially in the long term. This is particularly pertinent in the health domain where recent advances in medicine have made the long-term consequences of health-related misbehavior more, not less important. This is partly because the elimination of infectious diseases has increased life expectancy, so that minor dysfunctions have more time to develop into chronic diseases.

## III. ILLUSIONS OF CONTROL IN OBESITY TREATMENT & PREVENTION

How much control a person has over weight in particular is one of the most pressing questions in the weight-control area. The wellness movement, which has taken hold of the health mentality of the U.S. population, is rooted in the concept of personal control over health. This focus on individual responsibility reaches extremes in the search for the perfect body, where "… self-management, hard work, delay of gratification, and impulse control are qualities projected on people with the right body. People with the wrong bodies, those overweight and unfit, are thought to be indulgent, lazy, and lacking control" [8].

Control over our bodies must, of course, be considered within the context of biological realities. Unfortunately, obesity/weight control is an area in which biological realities collide head on with cultural pressures [8]. And where cultural pressures win. One consequence is that individuals (as does the culture in general) assume more control than actually exists.

In the remainder of this section, I distinguish between two notions of control: personal control which implies the belief of "being in control of things," and the belief that "things are being under control," which implies a meaningful ordered situation. I will also discuss how the former affects treatment, while the latter affects prevention.

### III.1 Misconceptions about Treatment: Illusion of Voluntary Control over Energy Intake and Expenditure

As already mentioned, most obese individuals attempting to lose weight do so themselves without seeking professional help [9]. For example, dieting, the mainstay of obesity treatment is most often undertaken as a self-directed process with instruction from a book or slimming club within the community, or often just by

self-induced restraint. There are at least two reasons for this. First, given the sheer number of obese individuals who need help, it is clear that there aren't enough health professionals available to provide intensive, long-term treatment.

Second, both the nature of the problem as well as its solution seem on the surface to be transparent enough. Figure 1 depicts the widely shared mental model of the problem and its solution ([19, p. 617], [34, p. 231]). Body weight is viewed as being dependent on the balance between energy intake (EI) and energy expenditure (EE), both of which are assumed to be under *voluntary control.* Unbalancing the energy balance equation in the direction of weight loss would, therefore, appear to be straightforward: reduce daily caloric intake below the daily energy requirements, increase energy expenditure (through increased physical activity for example), or do both.

The notion that people are in full control of their energy intake and expenditure levels is so widely held that most people believe that every overweight person can achieve slenderness and should pursue that goal. It is also why obese people are stereotyped as lacking in self-control, and why many are often scolded by parents and friends for lacking the discipline to resolve their weight problems [34, p. 260].

In reality, unbalancing the energy balance equation in the direction of weight loss has proven not to be that straightforward. Indeed,

> … one should be warned, if the concept of fat balance is exceedingly simple, in practice, there are certain complexities. The complexities come about because our bodies cannot leave energy metabolism to chance; lack of energy means failure to thrive, and often, failure to survive. Thus it is an oversimplification to say that fat balance is just a matter of the difference between what is consumed and stored, versus what is mobilized and oxidized. This is true, but it shall turn out that one must also consider the body's ability to regulate the rate at which all energy-yielding nutrients are oxidized, and to alter the proportions according to a logic that is part of our ancestral heritage [13, p. 219].

Specifically, the simplistic open-loop mental model of Figure 1 fails to capture some key *homeostatic mechanisms* that are key to understanding the etiology of obesity and its treatment. In the remainder of this section I discuss those mechanisms that are key to understanding why EI and EE may not be entirely under personal control.

***The Body's Adaptation to Energy Restriction (and the Regulation of EE)***.

The body's total energy expenditure can be divided conceptually into three components. The largest component is the resting energy expenditure (REE), defined as the energy expended to sustain the basic

metabolic functions of an awaken individual at rest. In most sedentary adults, the REE makes up about 60 to 70% of total energy expenditure.

The second largest component of daily energy expenditure is the energy expended for muscular work, known as the thermic effect of activity (TEA). The TEA of an individual not engaged in heavy labor accounts for 15 to 20% of daily energy expenditure, but can increase by a factor of 2 or more with very heavy exercise [34, p. 240].

The third component of energy expenditure is the thermic effect of food (TEF). It constitutes the metabolic costs of processing a meal, which include the costs of digestion, absorption, transport, and storage. The TEF accounts for approximately 10% of the daily energy expenditure, but can vary depending on the amount and the composition of the diet [34, p. 239].

Weight loss induces involuntary declines in both the thermic effect of food (TEF) and the resting energy expenditure (REE). Since TEF constitutes the metabolic costs of processing a meal, eating less while on a diet would naturally lower the thermic effect of food. This effect is depicted as link (1) in Figure 2. The drop however is not very significant in absolute terms since TEF accounts for only 10% of the daily energy expenditure.

On the other hand, the impact of a decrease in body weight on REE is more substantial. As lean body tissue (the principal metabolically active component of total body mass) is shed during weight loss, REE falls accordingly. This is captured by link (2) between "Body Mass & Energy Stores" and REE in Figure 2. As the body's REE drops, the size of the energy deficit effectively shrinks, which in turn slows subsequent losses in body weight. Because resting energy expenditure accounts for 60-70% of total energy expenditure, the drop in REE can significantly alter the body's energy balance and, in turn, the success of weight reduction efforts.

Unfortunately for the dieter, additional homeostatic adaptations occur during food energy restriction that cause REE to decline by an amount greater than can be accounted for by the losses in lean body tissue. Link (3) in Figure 2 represents the body's physiological adaptation to conserve energy during caloric deprivation in order to restrain the rate of tissue loss. This is achieved chiefly through hormonal mechanisms that operate to decrease the metabolic activity at the cellular level, in essence enhancing the tissues' metabolic efficiency [27]. This homeostatic mechanism provides a buffer against energy imbalance and hence spares both lean and fat

tissue compartments. The survival value of such an energy-sparing regulatory process that aims to limit tissue depletion during food scarcity is obvious.

For the dieter, this energy conserving adaptation causes the diet to become progressively less effective. As a result, weight loss plateaus at a level considerably less than predicted from the mathematics of the static energy equation. It also explains why it is difficult to prevent the frequently observed recidivism of obesity in obese subjects who lost weight. Formerly-obese persons may require 10-15% fewer calories to maintain the newly reached "normal" body weight than a person who has never been obese and who is therefore not experiencing a diet-induced depression in REE [16].

***The Body's Adaptation to Weight Loss (and the Regulation of EI)***.

Perhaps even more discouraging, is the difficulty most dieters experience to *maintain* these "disappointingly" small weight losses. A review of the literature on weight loss reveals that a majority of patients who succeed in losing weight eventually regain it [23]. Typically, one- to two-thirds of the lost weight is regained within a year, and almost all of it within 5 years [19, pp. 617-618].

Such statistics suggest that while one can crash off large amounts of weight in a relatively short time by simply not eating, this success is short-lived, and eventually the urge to eat wins out and the weight is regained [19, pp. 617-618]. The reason is that EI is not totally under self-control as we would like to think. Instead, it is regulated by the body's own fat tissue (links 4,5 in Figure 2).

The amount of fat in adipose tissue is the cumulative sum over time of the differences between energy intake and energy expenditure. It has become clear in recent years that the adipocyte is not the passive receptacle of fat that was once thought. It secretes a number of active substances that have an impact on metabolic/physiologic functions elsewhere in the body. One such substance is leptin, which is synthesized in fat cells and secreted into the blood stream in concentrations that are proportional to total adipose stores. Recent research findings suggest that leptin plays an important role in the body's weight-regulating system as the main signal that lets the brain know how much fat the body has stored. When fat stores rise, adipocytes produce leptin, and the elevated concentration of the hormone in turn signals the brain that it's time to stop eating and increase activity levels. Conversely, when fat stores decline, leptin concentrations go down too, and that signals the brain to try to counteract the weight loss with increased feeding and lowered activity ([24, p. 132); [23]). Obese people, thus, do not get any help from their adipose tissue to reduce their appetites. Quite the opposite;

initial weight losses, lead to a decrease in fat cell size, which trigger compensatory adjustments such as increased hunger and caloric intake which, in turn, lead to weight regain.

Figure 2 only begins to illustrate how the real causal sequence underlying human eating behavior/obesity involves a complicated set of physiologic feedback mechanisms. However, most people (and patients) don't see this. Instead, people readily accept a far easier explanation of their weight regulation/eating behavior: that they control it.

> We can't possibly know (let alone keep track of) the tremendous number of (physiologic) influences on our behavior because we inhabit an extraordinarily complicated machine. So we develop a shorthand, a belief in the causal efficacy of our conscious thoughts. We believe in the magic of our own causal agency [32, p. 15].

### III.2 Misconceptions about Vulnerability: Illusion of Involuntary Maintenance of Body Weight

Humans have learned to rely on the body's self-regulatory mechanisms to maintain its internal environment. For example, body core temperature, body fluid volume and tonicity, blood glucose, as well as numerous other physiological conditions remain at characteristic levels throughout the many changes that occur daily and seasonally in the external environment. In feeding, humans have learned to regulate behavior in accordance with the body's biological states of need: to eat when hungry (when the body senses an energy deficit), and to stop eating when feeling full (when energy depots are replenished). And throughout most of human history, obesity was never a common health problem. It is, therefore, quite understandable for people to assume that a regulatory system exists in humans for maintaining body weight at some "natural" level. Such a system would be symmetrical, defending against both positive and negative energy balances that threaten to cause weight change. The underlying mental model is depicted in Figure 3.

Unfortunately, this is another illusion of control (in this case involuntary or automatic control) that may be seriously undermining obesity prevention efforts. In reality, humans display a system of weight regulation that is *asymmetrical.* Indeed, "Evolution has selected our physiology and behavior to favor over-consumption rather than under-consumption. This means that the system is more responsive to deficits in energy and nutrients than to increments" [4].

The interaction between a species and its food supply is one of the most important influences affecting genetic selection and adaptation. During our species' long evolutionary history as hunter-gatherers, energy

expenditure was high and food was scarce (or periodically scarce). Not surprisingly, food scarcity has been a powerful agent of natural selection for both our genes and cultural traits.

> Given the frequency of food shortages under natural conditions, selection favored individuals who could effectively store calories in times of surplus. For most individuals, such fat stores would be called on at least every 2-3 years. In this evolutionary context the usual range of human metabolic variation would have produced many individuals with a predisposition to become obese, yet they would never have the opportunity to do so. Furthermore, in this context there would be little or no natural selection *against* such a tendency. Selection could not provide for the eventuality of continuous surplus because it had simply never existed [6].

Furthermore, genetic traits that cause fatness improved the chances of survival in the face of food scarcities. Malnutrition from food shortages has a synergistic effect on infectious disease mortality, and decreases birth weights and rates of child growth as well. Thus, females with greater energy reserves in fat would have a selective advantage over their lean counterparts in withstanding the stress of food shortage, not only for themselves but for their fetuses or nursing children as well [7].

The remainder of this section discusses some of the homeostatic mechanisms that underlie the body's asymmetric system of weight regulation.

***The Body's Defenses against Weight Loss/Gain.***

Feeding is controlled by two regulatory subsystems, one short- and the other long-term as shown in Figure 4. The *short-term* component controls the onset and cessation of feeding on a meal-related basis. During a course of a meal, the presence of food in the gastrointestinal (GI) tract causes receptors in the stomach to stretch, while nutrients in the small intestine trigger the release of GI hormones (such as cholecystokinin). Together, gastric distension, nutrients in the small intestine, and GI hormones send messages about the amount of food eaten and the kinds of nutrients received to the brain (the hypothalamus). Ultimately, hunger diminishes as satiation occurs and people stop eating [34, p. 233].

Feeding provides energy substrate to meet the immediate metabolic needs of the body, the unused portion of which is stored primarily in depot fat reserves. The *long-term* regulatory component monitors the depletion/repletion of these reserves. As mentioned earlier, the body's fat reserves generate neuronal and endocrine factors that are reliable correlates of fat cell size. These factors serve to enhance or decrease the potency of the short-term satiety signals to the brain. That is, the sensitivity of the brain to the meal-generated satiety signals is determined in part by the size of the adipose mass so that "… an individual who has recently eaten insufficient food to maintain its weight will be less sensitive to meal-ending signals and, given the

opportunity, will consume larger meals on the average. Analogously, an individual who has enjoyed excess food and consequently gained some weight will, over time, become more sensitive to meal-terminating signals" [36].

The system's asymmetry is achieved in two ways. First, the neuronal and endocrine factors generated when fat cell size decreases (as a result of a negative energy balance) and that stimulate feeding are more potent than are the inhibitory signals generated when fat cell size increases (as a result of over-feeding) (link 5 in Figure 2). This was demonstrated in a series of human experiments conducted to assess if and how humans compensate for surreptitious dilutions and supplements to the energy value of their diet [20]. Under conditions of unlimited access to food, humans were found to readily compensate for caloric dilution, but were more tolerant of increases in caloric intake. For example, Mattes et al [20] found that when free-living subjects were provided lunches containing ~ 66% less calories than their customary midday meal, the subjects compensated for the lunch-time deficit by ingesting additional *nonlunch* calories. As a result, the total energy intakes did not decrease. In contrast, when the subjects were (covertly) provided lunches containing ~ 66% more calories than their customary midday meal, the subjects did not adjust their nonlunch energy intake to compensate. As a result, total energy intake was significantly higher.

> Two principles may be deduced. Firstly, that biological processes exert a strong defense against under-eating which serves to protect the body from an energy (nutritional) deficit. Therefore, under-eating must normally be an active and deliberate process. Secondly, in general biological defenses against over-consumption are weak or inadequate. This means that over-eating my occur despite efforts of people to prevent it [5].

There is a second asymmetry in the regulation of body weight in humans: when body fat is shed during weight loss, the size of fat cells dwindles but not their number ([23]; [34, p. 255]). Recall that the primary form in which excess food energy is stored in the body is fat in the fat tissue. The amount of fat in a person's body reflects both the *number* and *size* of the fat cells. Fat cells expand in size until they reach their biologic upper limit. When the cells approach maximal or "peak" size, a process of adipocyte proliferation is initiated, ultimately increasing the body's total adipocyte number. Thus obesity develops when a person's fat cells increase in number, in size, or quite often both. Once fat cells are formed, however, the number seems to remain fixed even if weight is lost ([23]; [34, p. 255]).

In summary, then, the body's system for weight/feeding regulation is asymmetrical. Indeed, a symmetrical system probably would have led to extinction in a food scarce environment. Lower-than-average

food levels would have led to a drop in weight to below “target”. That's the downside. But an abundance of food would have exceeded the capacity of the body to store it, leading to a maximum weight equal only to the target weight. There is no upside to balance the downside. That obesity wasn’t a realistic possibility during our species' long evolutionary history in a food scarce environment was a result (not the goal) of such an asymmetrical regulatory system. On the other hand, the current epidemic of weight gain in many cultures is an understandable function of a changed relationship between biology and the environment. And like our biology, our mental models did not adapt as well – still reflecting knowledge that no longer applies.

## IV. METHOD

The persistence and pervasiveness of illusions (about control among other things) suggests that raw real-life experience is often not enough to correct people's mental models. This is particularly true in experience with and learning about complex systems (such as body weight regulation). Barriers to learning from raw real-life experience include: imperfect information, confounding and ambiguous variables, and time delays between taking decision and its effects on the state of the system. And so, deficient mental models endure while people jump from one weight loss “silver bullet” to another.

When experimentation in real systems is infeasible or undesirable (which is certainly the case with disease), computer simulation becomes the main, and often the only, way people can discover for themselves how complex systems work [28, p. 38]. Simulation-based learning environments (also referred to as *Microworlds*), provide high-quality outcome feedback that is immediate, undistorted, and complete and so address many of the barriers to learning mentioned above.

Unlike a mental model, a computer simulator can reliably and efficiently trace through time the implications of a messy maze of interactions. Experience from working with human subjects in many domains indicates that even when people learn to comprehend the detailed relationships and interactions in complex systems, they are usually unable to determine accurately the *dynamic behavior* implied by these relationships. That is, being able to mentally "run" the model to figure out the dynamic implications over time is a much more difficult task.

A major part of the research effort discussed in this paper was devoted to the development of an integrative System Dynamics computer model that aims to structure and clarify the complex web of

dynamically interacting mechanisms that regulate human metabolism and body weight. An overview of the model's four major subsystems and some of their interrelationships is provided in Figure 5. The model's four inter-related subsystems are: Energy Intake (EI); Energy Expenditure (EE); Energy Metabolism; and Body Composition. The model seeks to integrate the processes of human metabolism, hormonal regulation, body composition, nutrition, and physical activity. These processes are typically fragmented between many different disciplines and conceptual frameworks. This work, thus, seeks to bring these ideas together highlighting the interdependence of these various aspects of the complex system that is the human body. A detailed description of the model's structure, its mathematical formulation, and its validation is provided in [1].

The complex physiologic/metabolic processes regulating human energy and body weight and composition are modeled using the feedback principles of the *System Dynamics* approach.. While the words complex, dynamic, and system have been applied to all sorts of situations, *feedback* is the differentiating descriptor here. Indeed, feedback processes are seen in System Dynamics to hold the key to structuring and clarifying relationships within human systems and in understanding their dynamic behavior. Most succinctly, feedback is the transmission and return of information. More generally, feedback refers to the situation of X affecting Y and Y in turn affecting X perhaps through a chain of causes and effects. One cannot study the link between X and Y and, independently, the link between Y and X and predict how the system will behave. *Only the study of the whole system as a feedback system will lead to correct results*.

As shown in Figure 4, human feeding is regulated by two feedback systems, one short- and the other long-term. Perhaps less obvious is the set of interrelated feedback loops regulating energy intake and expenditure captured in Figure 2. For example, one can trace the following two interrelated loops: (1) A daily energy deficit → a drop in REE → a drop in daily energy expenditure → a decrease in the daily energy deficit; and (2) A daily energy deficit → a loss in body weight → a drop in REE → a drop in daily energy expenditure → a decrease in the daily energy deficit. In a dieting scenario, the two feedback loops reinforce one another. But, if the energy deficit were to be induced by an exercise treatment, the intervention could induce an increase in fat free mass (FFM), which could in turn elevate rather than lower REE. Human intuition (mental models) cannot reliably assess the resulting net effect of such interactions. Even though the dynamic implications of the isolated loops may be reasonably obvious, the interconnected feedback structure of even just two loops (not to mention the much higher complexity of the entire system) is often too complex for mental simulation. One

needs a formal system of “bookkeeping,” and the mathematical apparatus of a System Dynamics model provides such a system [2].

The above homeostatic mechanisms act by so-called *negative feedback*. Feedback loops divide naturally into two categories, which are labeled deviation-counteracting feedback or negative loops and deviation-amplifying feedback or positive loops. Negative loops counter and oppose change, while positive loops tend to reinforce or amplify whatever is happening in the system.

While most control systems of the body act by negative feedback, in rare instances, the body has learned to use positive feedback to its advantage. For example, in progressive training, an initial training overload causes physiological capacity to expand. As the physiological capacity of the body expands, the initial training stimulus must then be increased (progression) to maintain overload, leading to further expansion of physiological capacity. In other words, the initiating stimulus causes more of the same, which is *positive feedback*. (In all cases in which positive feedback is useful, the positive feedback itself is part of an overall negative process, which causes the change to eventually plateau.)

Ultimately, the purpose in applying System Dynamics is to facilitate understanding of and make predictions about the relationship between the behavior of a complex system over time and its underlying feedback structure. For this, system dynamicists rely on computer simulation. In the next section, I attempt to demonstrate the utility of this type of model to gain insight into the dynamics of body weight regulation. To do this, the model will serve as a laboratory for controlled experimentation.

**V. RESULTS**

Three experiments are presented which aim to demonstrate the model's potential utility as a learning environment for end-users (patients or potential patients), who are the primary decision makers in obesity treatment/prevention. Specifically, the experiments provide possible scenarios to allow learners to "discover" deficiencies in the simplistic mental models of weight regulation (Figures 1 and 3) which underlie the illusions of control discussed above. To build confidence in the model, the first experiment replicates experimental results from the literature. In a real learning environment, all following experiments would be conducted with the model tailored to each learner's personal parameters (in part, to mitigate definsive denials such as notions of

"it won't happen to me"). For the purposes of this paper, and in order to maintain a consistency between the three experiments, the subject's profile used in experiment 1 is maintained throughout.

### V.1 Impact of Dieting on Body weight and Energy Expenditure

Estimates of weight loss in response to food restriction go something like this: one kilogram of lost body tissue contains about 18.3 MJ[1]; a negative energy balance of 100 kcal per day (equivalent to 0.42 MJ) would, thus, shed about 8 kg in the first year and 80 kg over ten years. The assumption being that the body's energy requirements remain steady as body size decreases, and that there is a fixed energy cost of 18.3 MJ/kg of lost tissue [33].

It has already argued why in reality the weight loss achieved by food restriction is almost invariably less than that expected from the apparent caloric deficit. The first experiment sought to quantify the size of the gap between expected weight loss and that actually achieved. To do this, the conditions of an experiment conducted by Prentice et al [26] was replicated. In it, three healthy males (average weight 73.67 kg) lived in a metabolic suite for seven days during which they were fed a balanced diet to maintain energy balance. After the maintenance week, and while still living in the metabolic suite, the three subjects were underfed for 12 days. The diet was set at 3.5 MJ/day, which was approximately two thirds below the maintenance diet (inducing an average energy deficit of 8.9 MJ/d). The diet provided 31% of its energy as protein, 24% as fat, and 45% as carbohydrate.

Using the static energy balance equation (Figure 1) and assuming an energy density of body tissue of 18.3 MJ/kg, we would estimate a constant rate of weight loss of 0.48 kg/day, and a total of 5.8 kg for the 12-day period. This "estimate" is depicted in Figure 6 as curve 1. Curve 2 shows the observed changes in body weight of one of the three subjects[2] during the 19-day (456 hour) experiment, and the model's replication of the experimental treatment for the same subject is depicted as curve 3. At the end of the 12-day under-feeding period, the subject's weight dropped from 77 to 73 kg (a –5.2% change). As shown in the figure, the model closely replicated the actual results (showing a weight drop of 4.6 kg).

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[1] The Forbes [10, p. 216] empirical relationship between changes in FFM and FM permits one to predict the composition of weight loss/gain in humans. For an 75 kg male with 15% body fat, lost weight will be approximately 50% FM and 50% FFM. The energy content of fat is 0.032 MJ/gm [34, p. 142], and that of pure protein is 0.017 MJ/gm [19, p. 85]. Using the most commonly used hydration constant for FFM of 0.73 (i.e., assuming FFM to being 73% water and 27% protein) one arrives at the energy densities of FFM and FM to be 4.6 MJ/kg and 32 MJ/kg respectively. Thus, one kg of body tissue (50% FM, 50% FFM) has an energy content of 18.3 MJ.

In addition to shedding weight, the subject's body adapted to the energy deficit by lowering the maintenance energy expenditure. As shown in Figure 7, resting energy expenditure (REE) decreased by 0.8 MJ (a 11% drop) during the 12-day period. Notice that the decrease was proportionately greater than the decrease in body-weight: 11% v. 5.2 %. As shown in Figure 7, both in the human subject and in the model, REE responded almost immediately and progressively to under-feeding (which commenced on day 8, after one week on the maintenance diet).

The results demonstrate that change in body weight is not the only mechanism by which the body accommodates energy imbalance. Decreases in body weight were accompanied by continuous changes in the body's maintenance energy requirements, which induced a drop in REE. Because resting energy expenditure accounts for 60-70% of total energy expenditure, the drop in REE significantly alters the body's energy balance and, in turn, the amount of weight lost. As a result, weight loss was significantly less than that expected from the apparent caloric deficit.

The results demonstrate how energy balance is not entirely under voluntary control (as implied by the static open-loop model of Figure 1); rather the regulation of energy balance is significantly influenced by involuntary metabolic/physiologic adaptations that regulate energy expenditure. Ignoring these physiologic adaptations may sell more diet books but will inevitably lead to spurious predictions of treatment outcomes. Which should be of concern, because patients' expectations about treatment outcomes and the degree to which they are met are likely to affect self-efficacy and relapse [11].

**V.2 Post-Dieting Food-Intake Regulation**

The purpose of the second experiment (not replicated in the Prentice et al study) was to assess the body's self-regulating capacity to compensate for weight loss by inducing higher levels of energy intake. To accomplish this, the simulation was extended for a third phase in which food intake was ad libitum. The length of the third phase was 554 hours, making it approximately equal to the combined maintenance and underfeeding phases, for a total run of 1,000 hours (approximately 6 weeks).

Figure 8 shows the changes in body weight throughout the entire 1,000-hour period: living on a maintenance diet, under-feeding, and free feeding. Notice that early in the free-feeding period, weight continued

[2] Subject number 1603 was selected randomly. His initial weight was 77 kg. The results of all three subjects were comparable.

to drop. This is because of the natural delay involved in boosting the level of one's diet from a severely low (3.5 MJ/day) to a higher level. During the ramping up period, daily food intake levels progressively increase, but initially would remain below the "maintenance" level diet. As a result weight continues to decline. Eventually, though, daily food intake rises above the maintenance level (see Figure 9), causing weight to first stabilize, and then start increasing. By the end of the six-week period (and 23 days after the beginning of the free-feeding phase) weight had increased to 75 kg.

The results of the experiment demonstrate that the problem of recidivism is caused in part by the fact that energy intake (EI) is not solely under self-control as most of us would like to think. Specifically, the results indicate that after a period of underfeeding, voluntary energy intake progressively increases, plateauing at a level above that consumed during weight maintenance, inducing a rapid repletion of lost weight. Obese people, thus, do not get any help from their adipose tissue to reduce their appetites. Quite the opposite; initial weight losses, lead to a decrease in fat cell size, which trigger compensatory adjustments such as increased hunger and caloric intake which, in turn, lead to weight regain.

This perhaps explains why people who try to lose weight become trapped in weight cycling, the endless repeating rounds of weight loss and regain. *Yo-yo dieting* became the colloquial term for this process. Recently there has been growing concern that repeated weight loss followed by weight gain may have detrimental health, metabolic, and psychological consequences [8].

### V.3 Asymmetry in the Body's Feeding/Weight Regulation

In the past, it was generally accepted that the obese condition was simply a problem of gluttony. More and more, the evidence is indicating that excess weight is the result of reduced physical activity rather than increased caloric intake [19, p. 623]. Today, in the United States energy expenditure for most adults rarely climbs above the resting level, with walking being the most prevalent form of physical activity. Economic “incentives” seem to be driving the trend. In recent years the cost of burning extra calories has surged as work has become increasingly more sedentary. "To put it simply, it used to be that you got paid to exercise… If you wanted an income, you engaged in manual labor. Today, you pay to exercise, not so much in money, but in foregone leisure time" [25]. The purpose of the third experiment was to assess the behavior of the body's asymmetric weight regulation system in the current environment of low energy expenditure and abundant food, and its impact on body weight.

According to World Health Organization (WHO) reference standards, the energy cost of routine physical activity is determined as the product of the REE and an appropriate activity factor [33]. This is implemented in the model using the *physical activity ratio (PAR)*. Total 24-hour energy expenditure (EE) would, thus, be equal to the REE multiplied by the physical activity factor (PAR) and added to the energy cost of diet-induced thermogenesis. For free-living conditions and a moderate level of physical activity, an average PAR value of 1.4 would be typical ([33], [19, p. 154]).

To maintain consistency with the first two experiments, the subject of the simulation was chosen to be a male with an initial body weight of 75 kg and 15% body fat. Assuming a PAR of 1.4, this body weight is maintained on a balanced daily dietary input of 12 MJ composed of 50% carbohydrate, 35% fat, and 15 % protein. In Figure 10, curve 1 shows the base case scenario in which weight is maintained at the 75 kg level. The length of the experiment was 2000 hours (approximately 12 weeks).

To assess the behavior of the body's asymmetric weight regulation system, two additional simulations were conducted: the first simulates a high-energy throughput environment, while the second simulates a more sedentary environment. Food intake was ad libitum in both cases (as in the base case scenario).

To simulate the high-energy throughput environment, the physical activity ratio (PAR) was increased from 1.4 to 1.8. The results of the simulation are depicted in Figures 10 and 11. Because of the natural delay involved in making a full adjustment in energy intake (from the daily maintenance diet of 12 MJ), the increased level of physical activity induces a negative energy balance. As a result, there is an initial drop in body weight (curve 2 in Figure 10). It doesn't take long, however, before dietary energy intake starts progressively increasing (Figure 11, curve 2). By week 9 (1,500 hours), all lost weight was regained, and body weight returns to its initial level. Food energy intake, however, stabilizes at a level above the initial maintenance diet in order to accommodate the new more strenuous level of physical activity.

On the other hand, when PAR was lowered to 1 (simulating a change to a more sedentary environment), weight increases and *remains* above the initial level (curve 3 in Figure 10). Initially body weight increases because of the natural delay involved in decreasing the level of one's diet to a lower level. In contrast to the high-energy scenario, the sustained positive energy balance induced here, in turn, induces a decrease in voluntary food intake (curve 3 in Figure 11). Notice, however, that the magnitude and speed of the adjustment is smaller and slower in comparison to the high-energy throughput scenario. As a result, the subject loses some

but not all the weight gained early on. At the end of the simulated 12 weeks, the subject maintains a weight gain of 2.5 kg.

The simulation results clearly demonstrate the asymmetry in the weight/feeding regulation system: rapid, accurate compensation for negative energy balance, but more tolerance for positive energy balance. Analogous, but not identical, results were reported in a study to assess the ability of human subjects to return to their initial weight levels after substantial weight loss. The experimental manipulation involved underfeeding the subjects as opposed to changing physical activity levels. All weight lost during the underfeeding period was rapidly regained as voluntary energy intake increased above the initial maintenance diet when the subjects were allowed to free feed. Particularly significant was the finding that none of the subjects reported consciously trying to regain weight lost during underfeeding [14].

## VI. DISCUSSION

Control *does* appear to be important to health. However, control over our bodies must be considered within the context of biological realities. In this study we distinguished between two notions of control: personal control which implies the belief of "being in control of things," and the belief that "things are being under control," which implies a meaningful ordered situation. The widely shared energy balance model of weight regulation (Figure 1) instills the former type of illusion. The results of the first two simulation experiments underscore the model's deficiencies, demonstrating how the regulation of energy balance is significantly influenced by *involuntary* metabolic/physiologic adaptations that regulate both energy expenditure and energy intake. Ignoring these physiologic adaptations to energy expenditure (EE) inevitably leads to spurious predictions of treatment outcomes. Which should be of concern, because patients' expectations about treatment outcomes and the degree to which they are met are likely to affect self-efficacy and relapse.

The lesson from the third experiment (which demonstrated the asymmetry of the body's weight-regulation processes) is simple: we need to replace the passive model of involuntary/automatic weight maintenance with an assertive model of cognitive control to proactively resist the "obesifying" aspects of the current environment. This, it could be argued, is a prerequisite to any strategy towards obesity prevention. While the attention to the treatment of obesity has heretofore overwhelmed that given to prevention, interest in obesity prevention is attracting increasing attention because of the growing realization that it may be

easier, less expensive, and more effective to change behavior so as to prevent weight gain or to reverse small gains, than to treat obesity after it has fully developed. A significant impediment to prevention, however, is an "automatic weight control" mentality that instills a false sense of invulnerability (the belief that “things are being under control”). If individuals believe that that they invulnerable then there is less motivation to protect themselves by taking direct action to decrease the probability of gaining weight.

***“Microworlds” for Double-Loop Learning.*** Because raw real-life experience is often not enough to correct people's mental models, deficient mental models endure while people jump from one weight loss “silver bullet” to another. The process whereby people learn to reach current goals in the context of their existing (and often deficient) mental models has been called “single-loop" learning. The term is borrowed from cybernetics where an error is detected and corrected without questioning or altering the underlying structure of the system (e.g., a thermostat is defined as a single-loop learner). By contrast, the goal of double-loop learning is to induce deep change in people's mental models creating more effective decision rules/strategies [28, p. 18].

An important role for the System Dynamics computer model presented in this paper is to serve as a laboratory for personal learning about obesity and weight management. Simulation-based learning environments (also referred to as microworlds) have been successfully applied in many environments (e.g., the military, pilot training flight simulators, power plant operations, etc.) to achieve double-loop learning because they address many of the barriers to learning from raw real-life experiences.

In the simulated environment of a microworld, feedback from actions/decisions is rapid and unambiguous. In real life, by contrast, the consequences of our actions are neither immediate nor unambiguous. When someone smokes a cigarette or eats a piece of chocolate cream pie, for example, not all of the consequences of doing so are evident in the moment the action is being taken. Yet all of these actions can have potentially serious longer-term consequences for that person's well being. A microworld allows time and space to be compressed or dilated, and thus time delays between actions/decisions and consequences (which in the case of human disease may be quite long) could be dramatically reduced. This could be of particularly effective when teaching for prevention. For example, the simulation-based learning environment can become a practical way to "experience" obesity-induced disease in advance of the real thing. This would allow patients (or potential patients) to see more clearly the long-term consequences of their lifestyle decisions, making them more perceptive of the slow, gradual physiologic changes that accompany weight gain.

In addition, microworlds provide high-quality outcome feedback. Because a System Dynamics model is a causal model, with clear cause-and-effect connections between modeled system variables, it is particularly suited to serve as a framework to explain/understand experimental observations (examples are provided in [2]). Furthermore, the controlled environment of a microworld allows the user to easily eliminate intruding outside variables. Real life offers no such control. Actions that cannot be reversed or taken back in real life can be redone countless times in the microworld. And of course, the learning experience can be highly "personalized," by tailoring model parameters to individuals. This can be particularly effective in softening a heretofore stubborn barrier to many a prevention message: the illusion of *unrealistic optimism* (the *unique invulnerability* or "it won't happen to me" notion*)* about health that is so pervasive and enduring [29].

Lay people have learned to rely on computer-based tools to handle the complexities of managing their portfolios, preparing their taxes, and even shopping for and making airline reservations. In this research effort we, hope to demonstrate both the feasibility and utility to leverage computer technology to support personal learning as well as health-related decision-making. Paradoxically, the recent advances in medicine have made the task of personal health management more, not less complex. This is partly because the elimination of infectious diseases has increased life expectancy, so that minor dysfunctions have more time to develop into chronic diseases.

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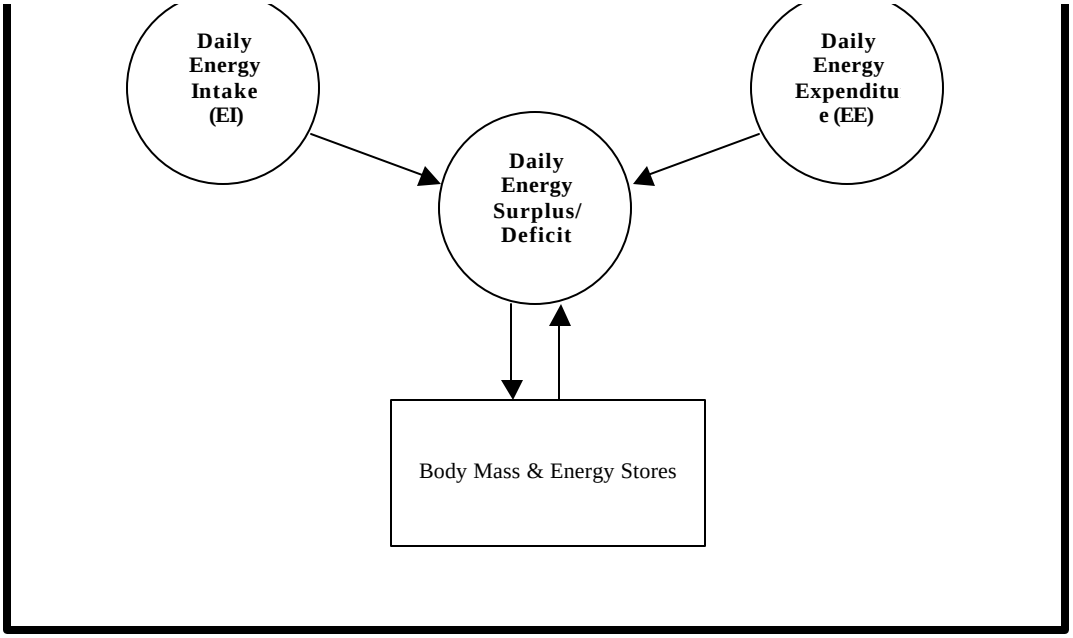


Figure 1

Energy Balance Equation

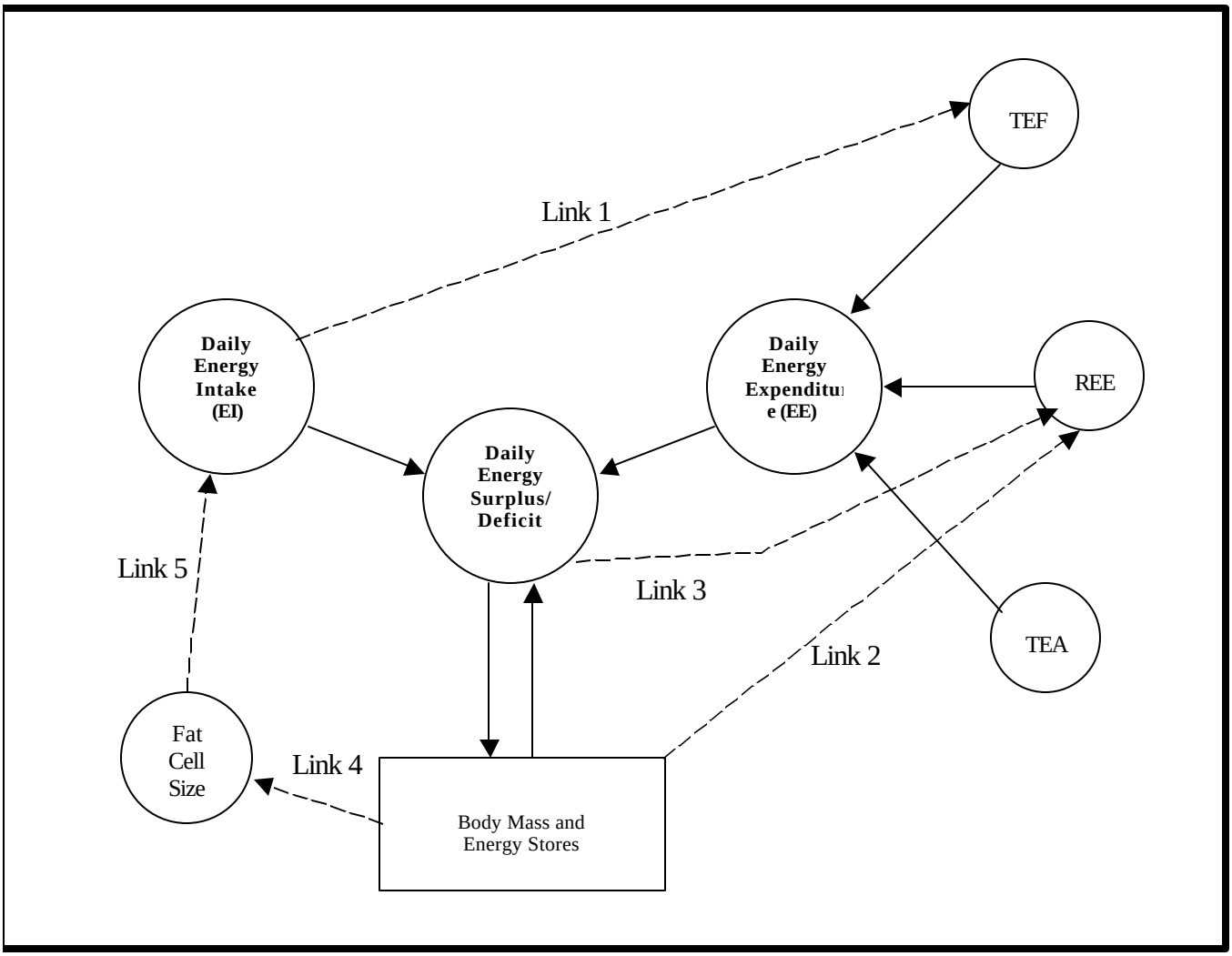


Figure 2

Added Links to Energy Balance Equation

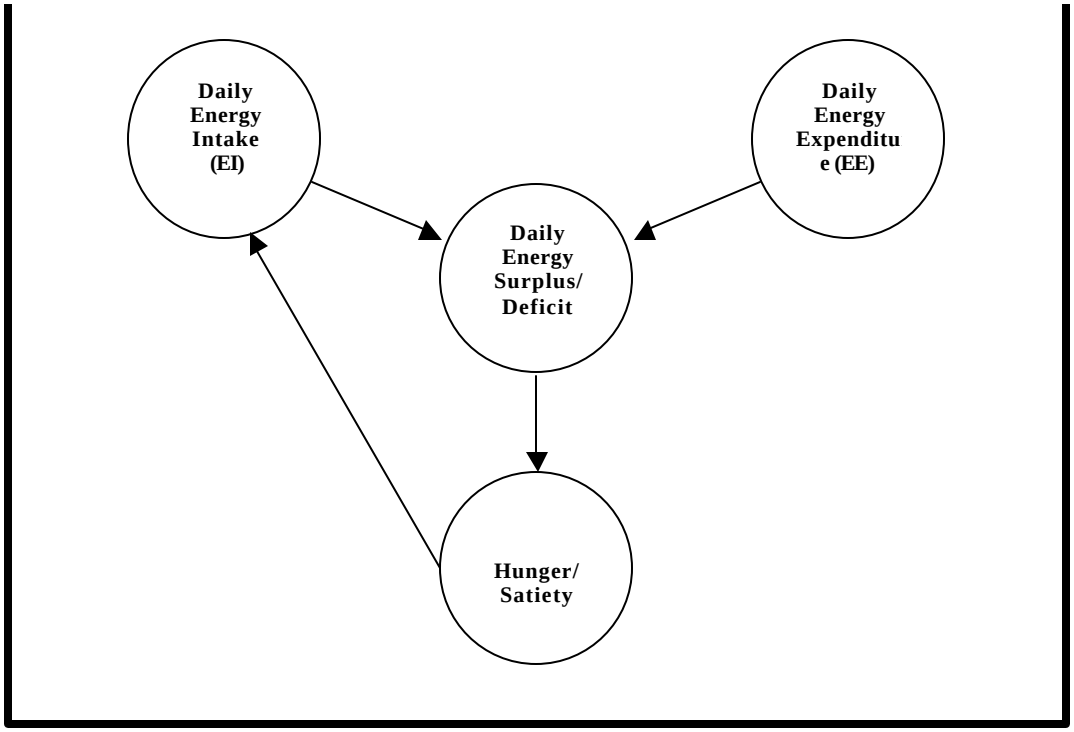


Figure 3

Weight Maintenance

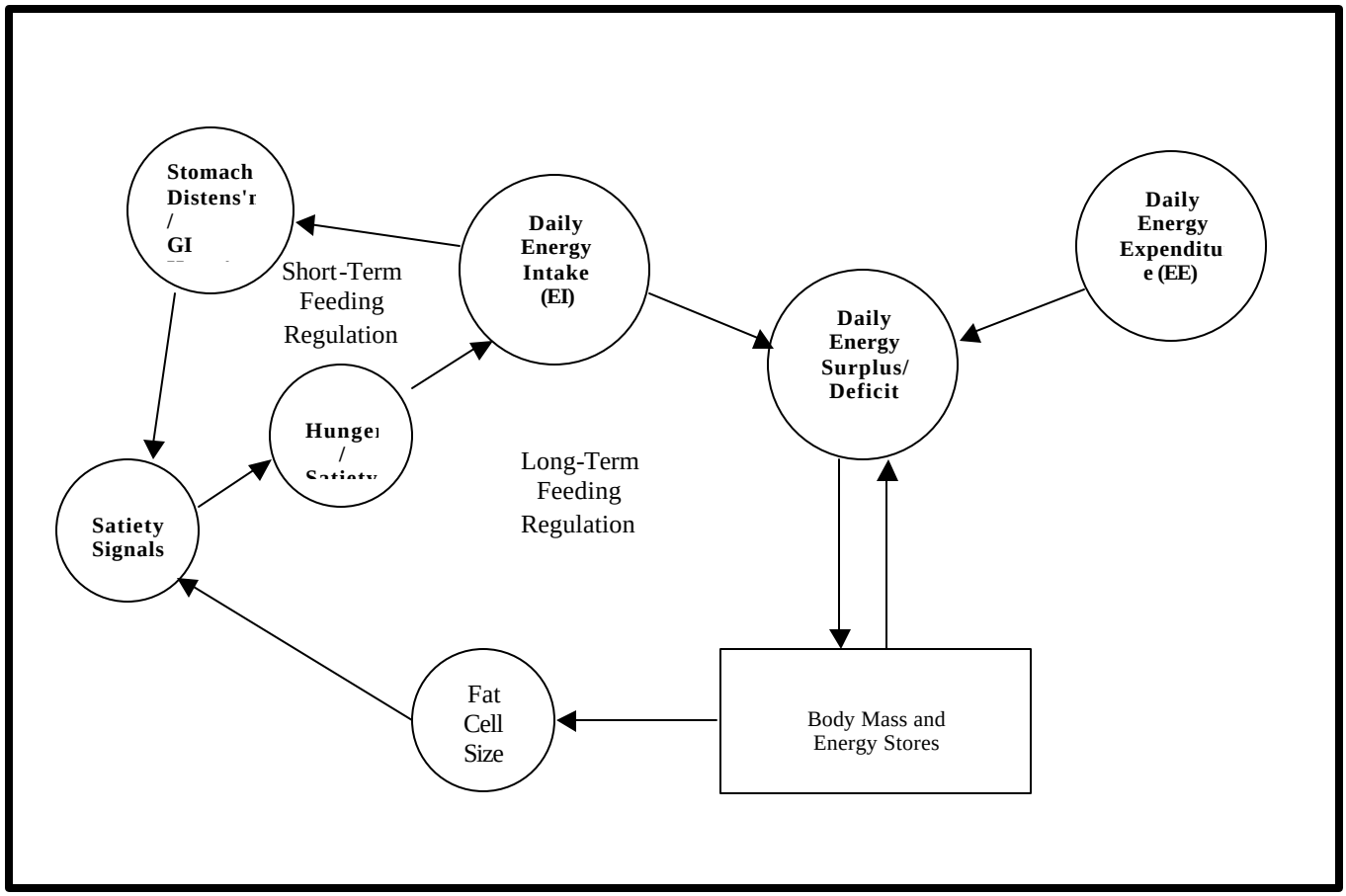


Figure 4

Short- and Long-Term Feeding Regulation

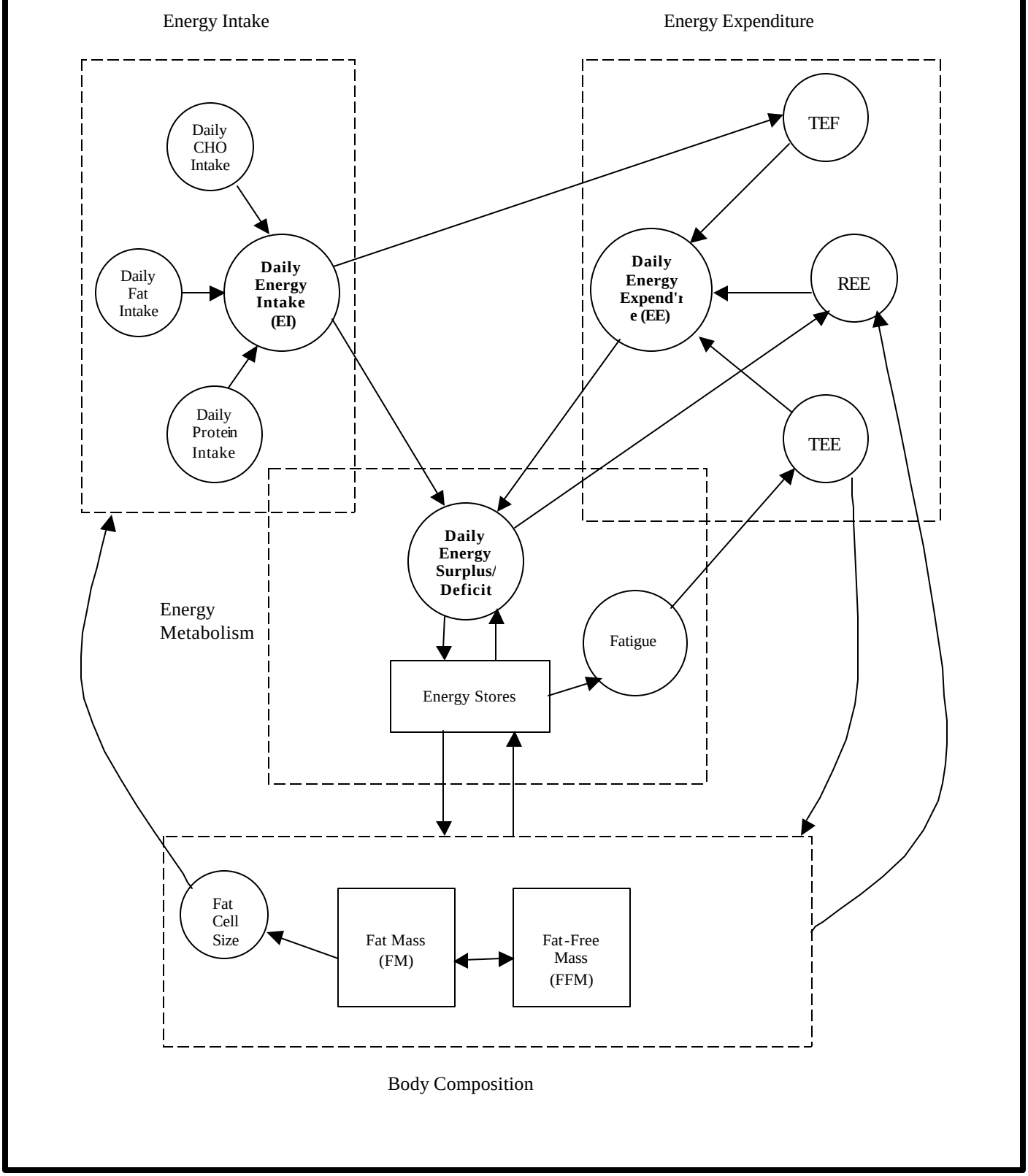


Figure 5

Overview of the Model's Four Subsystems

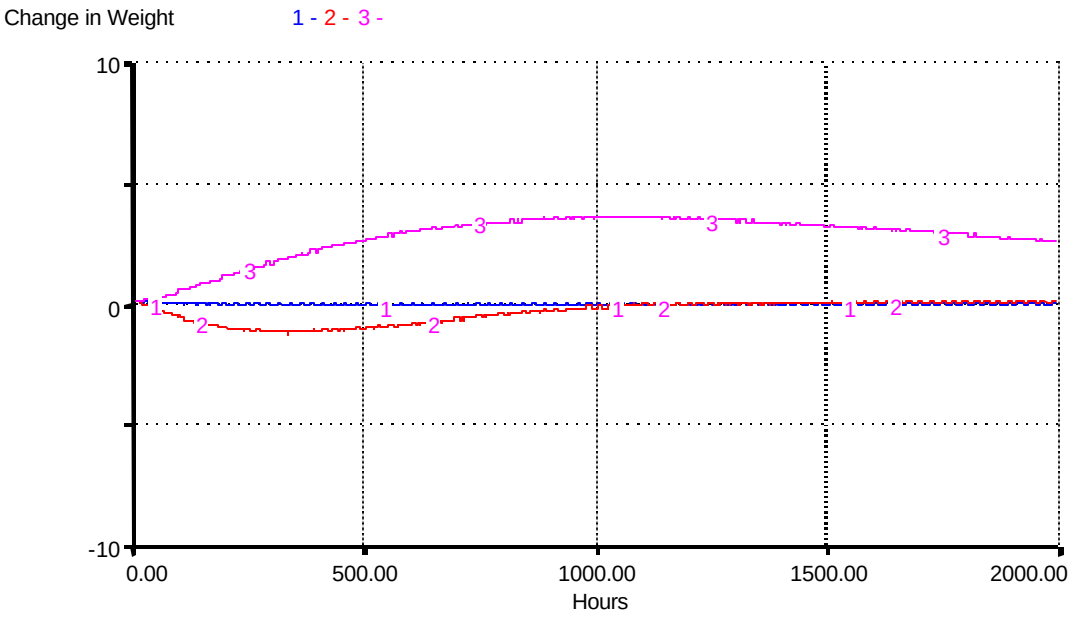


***Figure 10***

Change in weight

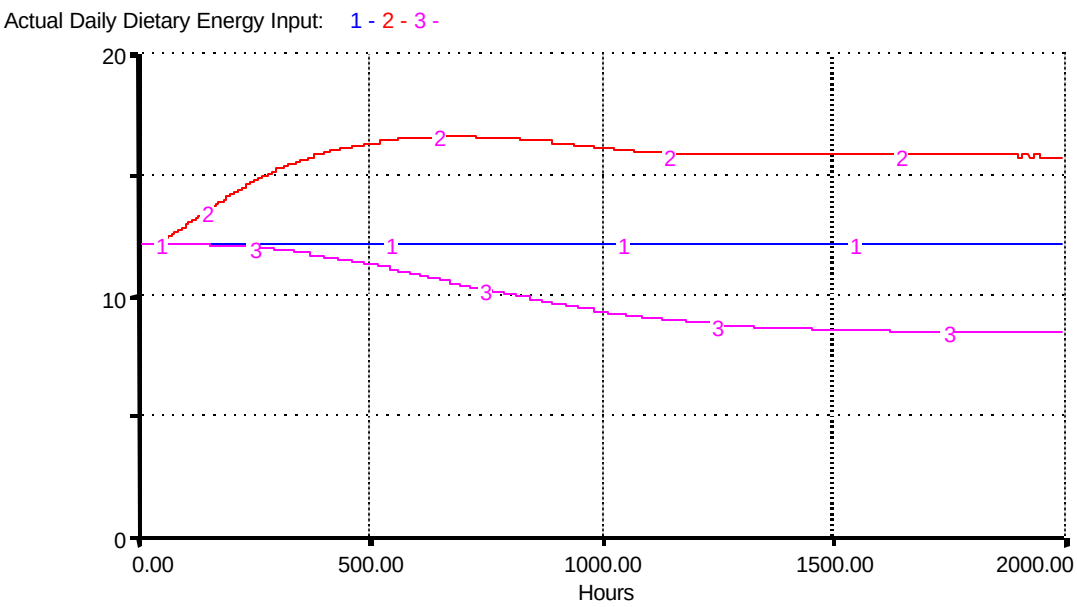


***Figure 11***

Adjustments in Food Energy Intake

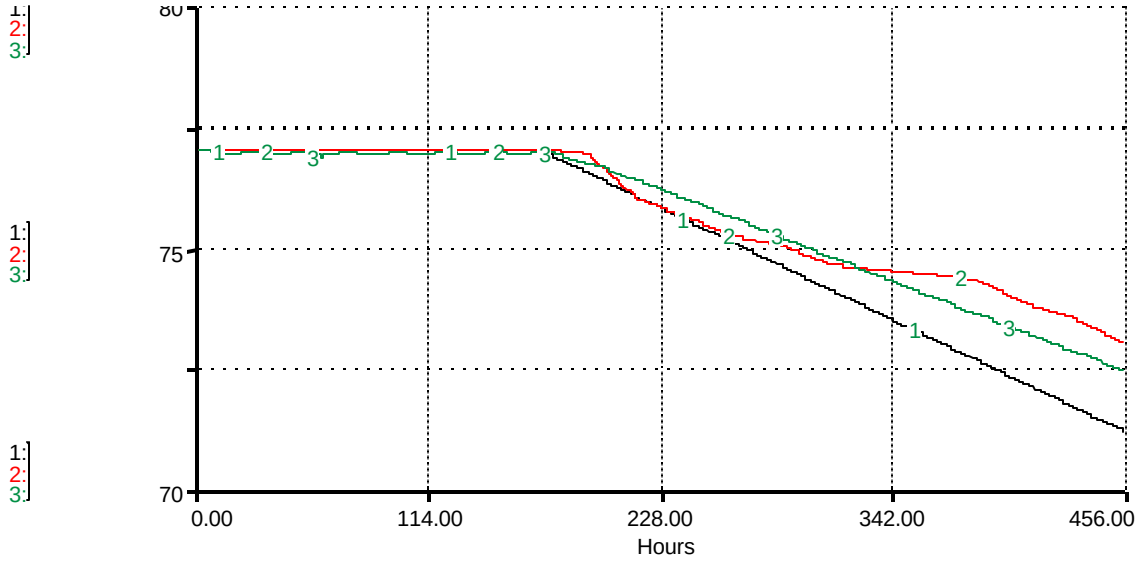


***Figure 6***

Weight Loss (in kg) due to Underfeeding

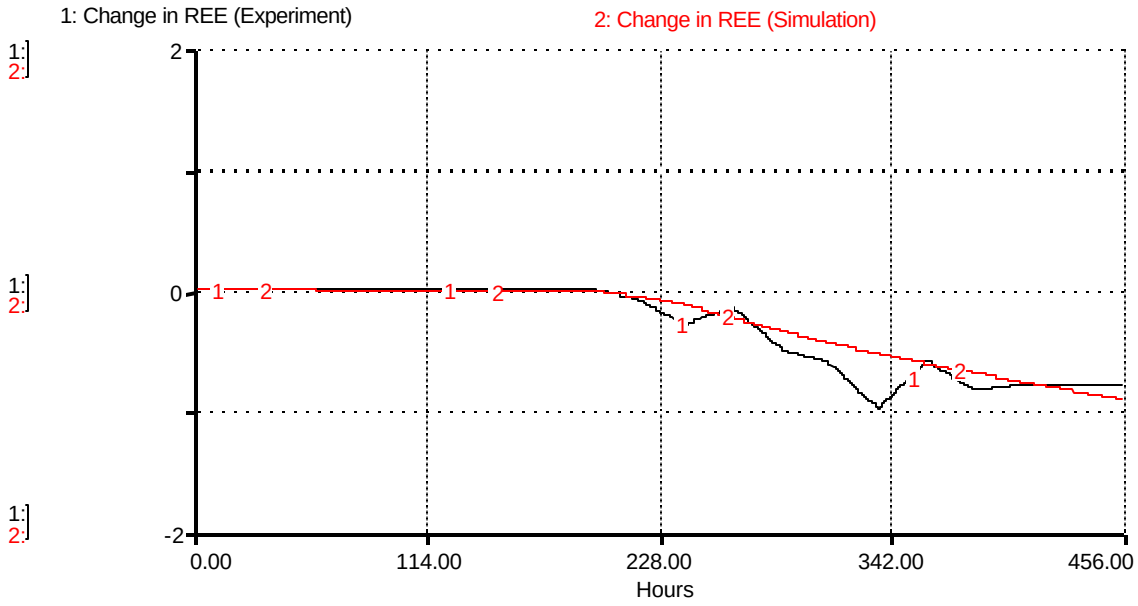


***Figure7***

Changes in REE (MJ)

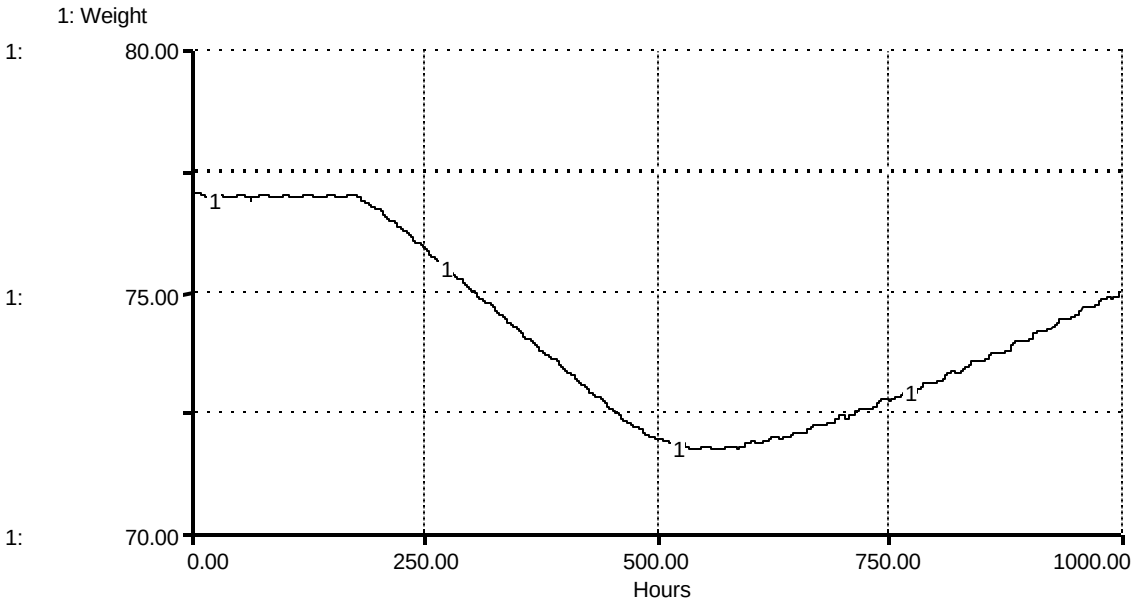


***Figure 8***

Weight Loss and Regain (kg)

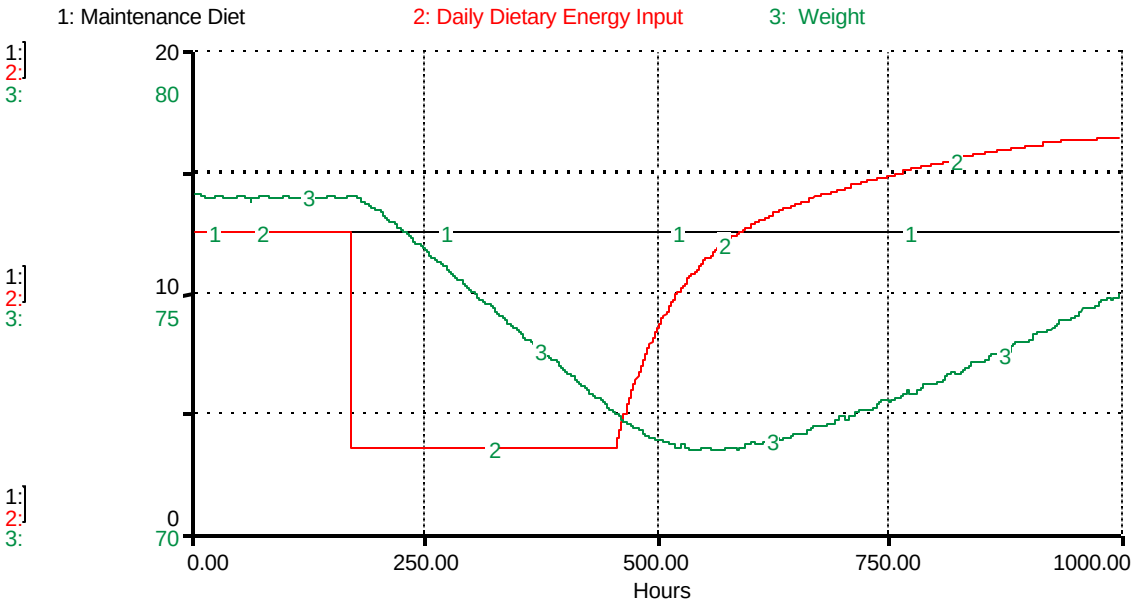


***Figure 9***

Daily Energy Intake (MJ)



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